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Document Number:

33) IV-D-62

Docket Number:

A-91-46

P.2
A-91-46
IV-D-62

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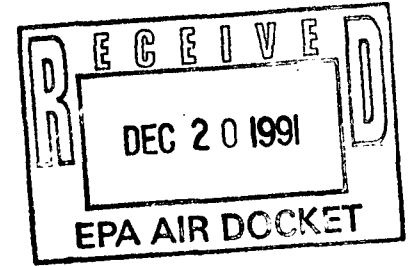
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December 20, 1991

BY HAND

Ms. Mary T. Smith
Director
Field Operations and Support Division
Office of Mobile Sources
EN-397F
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460



Re: Public Docket No. A-91-46

Dear Ms. Smith:

On the issue of public health as it relates to Ethyl Corporation's ("Ethyl") waiver application for use of HiTEC 3000 ("the Additive"), we are enclosing (Attachment 1) the EPA staff memorandum that discusses the basis for the Agency's decision in 1985 not to regulate manganese as a hazardous air pollutant under § 112 of the Clean Air Act.

This memorandum (entitled "Preliminary Analysis of the Health Effects Associated with Manganese and Concentrations Predicted to be Present in the Ambient Air for Various Averaging Times") makes the following relevant observations about manganese:

- Manganese oxides are "not soluble in water; consequently, only a small amount of [manganese] inhaled is biologically available." See Tab A, "Overview" (emphasis added).
- The annual average airborne manganese exposure levels estimated by EPA in 1985 from major stationary sources far exceed the manganese exposure levels at issue in this proceeding (by up to an order of magnitude or more). Yet, EPA concluded that these higher exposures would be safe in terms of both respiratory and neurological effects. See pages 2-3, 5.

We have also enclosed a brief summary of the recent study completed by the National Research Council ("NRC") of the National Academy of Sciences which strongly recommends that the

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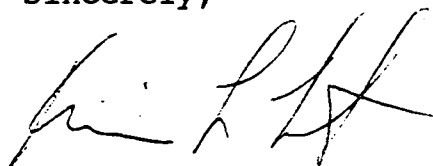
Ms. Mary T. Smith
December 20, 1991
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focus of programs to reduce ambient ozone concentrations should include more effective control of nitrogen oxide emissions (Attachment 2). To this end, Ethyl's 48-car test program showed that use of the Additive reduced nitrogen oxide emissions an average of 20 percent (0.11 gram per mile) for the test fleet over 75,000 miles of operation. Since mobile source emissions account for approximately 50 percent of the anthropogenic (manmade) emissions of nitrogen oxides, use of the Additive would provide a simple and cost-effective way to achieve substantial reductions in nitrogen oxide emissions -- reductions that would far outweigh even a slight increase in hydrocarbon emissions.^{1/}

Moreover, as Ethyl has explained in its submissions, use of the Additive by the refiner in commercial operation (as opposed to simply mixing the Additive with the base fuel as reflected in Ethyl's test program) would tend to eliminate the very small hydrocarbon emissions increase observed in the Ethyl test program as refiners use the Additive to replace aromatic octane in refinery operations.

The enclosures further demonstrate the significant benefits of the Additive and support a decision to approve Ethyl's waiver application.

Sincerely,



John J. Adams
F. William Brownell
Kevin L. Fast

Enclosures

cc: Public Docket A-91-46 (w/enclosures)
Mr. David C. Kortum (w/enclosures)
Mr. Carl Mazza (w/enclosures)
Dr. Peter Preuss (w/enclosures)

^{1/} See Systems Application International, Use of Urban Airshed Model to Assess the Effects of HiTEC 3000 Performance Additive on Urban Air Quality (May 1990) (A small hydrocarbon emission increase associated with use of the Additive would have no effect on ambient ozone concentrations, and together with the substantial reduction in nitrogen oxide emissions could actually result in reduced concentrations of ozone).



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

JUN 7 1985

A-84-19

II-B-3

MEMORANDUM

SUBJECT: Preliminary Analysis of the Health Effects Associated with Manganese and Concentrations Predicted to be Present in the Ambient Air for Various Averaging Times

FROM: Nancy B. Pate, DVM, MPH
Project Officer
Program Integration and Health Section, PAB

TO: The Files

Introduction

The purpose of this memorandum is to document for the record the analysis of the potential for ambient concentrations of manganese around major emission sources to approach concentrations for exposure periods which have resulted in identifiable adverse health effects in defined populations. The methodology used for this analysis involved four steps. First, based upon the information presented in the Health Assessment Document (HAD) for Manganese, the highest documented concentrations of manganese to which one could be exposed for several different exposure periods and not experience any identifiable adverse health effects were established. Secondly, the literature was reviewed to identify and characterize available ambient air quality data for manganese. In addition, limited site specific monitoring data for total suspended particulates were obtained. The third step involved the use of the Human Exposure Model (HEM) and the screening model outlined in the document "Procedures for Evaluating Air Quality Impact of New Stationary Sources" to estimate

ATTACHMENT 1

long-term and short-term average ambient concentrations, respectively, using the most current emissions data. The fourth and final step was to compare the available monitored data and modeling results against the concentration/exposure scenarios for manganese which have been selected as protective against adverse health effects associated with manganese.

Identification of Protective Levels

The HAD document reports neurotoxic and respiratory effects in animals and humans associated with exposure to manganese compounds or particulate matter containing manganese. A summary review of these studies is attached in TAB A. A sufficient number of human studies are available making it unnecessary to extrapolate from the animal data. The two major clinical manifestations associated with manganese emissions, i.e., neurotoxic and respiratory, are not a single biological end point but are relatively progressive given sufficient concentration and duration of exposure. Consequently, it is difficult to attempt to establish a precise "effect level."

The respiratory effects associated with inhalation exposure to manganese are identical to effects observed for any fine particulate matter ($\leq 2.5 \mu\text{m}$) whether or not it contains manganese. However, the neurotoxic effects are directly attributable to the amount of manganese that is ultimately absorbed into the blood stream and deposited in the brain. Consequently, as a conservative approach, based on information presented in the HAD, the highest documented concentrations of manganese were identified to which one could be exposed for defined exposure periods and not experience any clinically evident neurotoxic effects were identified. The HAD reports that there is no clear cut evidence of manganese neurotoxicity associated with

chronic exposure to manganese concentrations below $5000 \mu\text{g}/\text{m}^3$. However, for this analysis, the World Health Organization (WHO) 8-hour time weighted average of $300 \mu\text{g Mn}/\text{m}^3$ and the American Conference of Governmental Industrial Hygienists' (ACGIH) 15-minute maximum of $5000 \mu\text{g}/\text{m}^3$ were selected as protective levels for the neurotoxic effects associated with manganese. For respiratory effects, given that the effects are associated with particulate matter and are not dependent on the concentration of manganese in the particulate matter, the primary 24-hour and annual national ambient air quality standards (NAAQS) for particulate matter were identified as protective levels with which to compare particulate emissions from major manganese emission sources.

Ambient Air Quality Data

Nationwide air sampling data for particulate matter and the concentration of manganese in the particulate matter have been available and reported in the literature since the early 1950's. These data indicate that both particulate matter and manganese concentrations have continually declined. The arithmetic mean manganese concentration of urban air samples decreased from $0.11 \mu\text{g}/\text{m}^3$ in 1953-1957 to $0.073 \mu\text{g}/\text{m}^3$ in 1966-1967, and to $0.033 \mu\text{g}/\text{m}^3$ in 1982. Site specific monitoring data in the vicinity of several ferromanganese facilities are also available for particulate matter and/or manganese concentrations. Particulate emissions from the ferromanganese industry are documented as having the highest concentration of manganese compared to other major manganese emission sources (HAD, 1984; SAI, 1983). Monitoring data for 1965 and 1982 in the vicinity (downwind) of a major ferromanganese facility, show that TSP ranged from a 24-hour average around

300 $\mu\text{g}/\text{m}^3$ in 1965 to 20-48 $\mu\text{g}/\text{m}^3$ in 1982 with a geometric mean of 32 $\mu\text{g}/\text{m}^3$ (HAD, 1984). In 1983, there were only five ferroalloy facilities producing in the U.S. Site specific monitoring data were obtained for three of these facilities and are documented in TAB B. In all three cases, the data indicate that the NAAQS for particulate matter have been attained since 1981. Site specific monitoring was not readily available for other emission sources of manganese.

Air Quality Dispersion Modeling

Dispersion modeling was performed to estimate both annual and 1-hour concentrations. The HEM was used to estimate annual average ambient concentrations. The HEM combines a gaussian distribution model, emission release parameters and meteorological data to estimate annual concentrations within 20 miles of manganese emitting facilities (SAI, 1983). The dispersion methodology that was employed to estimate short-term (i.e., 1-hour) values is presented in TAB C. The HEM and the short-term model used emissions based on 1978 production and emission factors. The short-term model incorporated a safety factor to compensate for uncertainties in the model (the 1-hour modeled concentrations were doubled). The emission factors and total emissions of manganese using estimated available sources of information are presented in Table 1.

Comparison of Ambient Monitored and Modeled Data with Health Effects Data

The WHO 8-hour time weighted average of 300 $\mu\text{g Mn}/\text{m}^3$ and the ACGIH 15-minute maximum of 5000 $\mu\text{g Mn}/\text{m}^3$ are recommended to protect occupationally exposed personnel from the neurotoxic effects of manganese. Using uncontrolled emission estimates the short-term model indicated a possible concern from short-term emissions from the ferroalloy industry. The modeled concentrations

were then adjusted proportionally on the basis of current controlled emission data. The modeling results are presented in Table 2 along with the selected protective levels. This analysis reveals that current controls and production levels have lowered ambient manganese concentrations to levels below those selected as protective.

As pointed out previously, the respiratory effects elicited by particulate matter containing manganese are not attributed to the concentration of manganese in the particulate matter. Consequently, the primary NAAQS were selected as protective levels with an adequate margin of safety with which to compare ambient monitored particulate matter concentrations to assess the potential for these concentrations to elicit respiratory effects. Monitored TSP levels in the vicinity of three out of five ferroalloy facilities revealed that the standards are being attained. Since manganese exists as particulate matter and is reported to comprise a fraction of TSP matter that may range up to 25%, the most current modeled concentrations indicate that the NAAQS are being attained. Table 3 summarizes higher modeled manganese concentrations, monitored TSP concentration, and exposure levels recommended as protective.

Conclusions

Given the findings presented here, the manganese concentrations measured or estimated to be present in the ambient air even in the vicinity of major manganese emitting facilities are below those levels recommended as protective against the health effects associated with exposure to manganese emissions.

Table 1

Source Category	SAI Emission Factor ¹	Uncontrolled Emissions ² (Mg/yr)	Controlled Emissions (Mg/yr)
Power Plants	0.0055 kg Mn/Mg Coal Used	1488	2148 ³ [1.44]
Iron & Steel	0.02 kg Mn/Mg product	4178	1100 ³ [.26]
Ferroally	3.3 kg Mn/Mg product	5729	2584 ⁴ [.07]
Gray Iron Foundry	0.154 kg Mn/Mg product	2512	80 ³ [.03]
Coke Ovens	0.013 kg Mn/Mg Coal Used	884	360 ⁵ [.41]

¹Based on uncontrolled emission parameters as existed in 1978 (SAI, 1983).

²Based on 1978 production data (SAI, 1983).

³Based on 1981 production and 1981/82 control status (Radian, 1984).

⁴Based on ferroally production of 78,018 Mg in 1983 production (Minerals Yearbook, 1983) and uncontrolled emission factor (SAI 1983).

⁵Based on amount of coal coked in coke ovens (27,714,000 Mg in 1983) (AISI, 1984). Ccke ovens were not included by Radian.

*NOTE: Number in brackets represents the revised emissions as a proportion of the original emissions. This factor was used to proportionally adjust the modeled concentrations presented in Table 2.

Table 2

Sources Average Time	Estimated Exposure Levels ($\mu\text{g Mn}/\text{m}^3$)		Selected Protective Levels ($\mu\text{g Mn}/\text{m}^3$)
	Uncontrolled Emissions	Controlled Emissions	
Power Plants			
Annual	0.2	0.3	75 $\mu\text{g}/\text{m}^3$
24-hour	5	7	260 $\mu\text{g}/\text{m}^3$
8-hour	15	21	300 $\mu\text{g}/\text{m}^3$
15-minute	25	36	5000 $\mu\text{g}/\text{m}^3$
Iron and Steel Production			
Annual	10	2.6	75 $\mu\text{g}/\text{m}^3$
24-hour	128	33	260 $\mu\text{g}/\text{m}^3$
8-hour	350	91	300 $\mu\text{g}/\text{m}^3$
15 minute	500	130	5000 $\mu\text{g}/\text{m}^3$
Ferroalloy			
Annual	2.5	.13	75 $\mu\text{g}/\text{m}^3$
24-hour	900	45	260 $\mu\text{g}/\text{m}^3$
8-hour	2500	125	300 $\mu\text{g}/\text{m}^3$
15-minute	5000	250	5000 $\mu\text{g}/\text{m}^3$
Grey Iron Foundry			
Annual	0.5	0.02	75 $\mu\text{g}/\text{m}^3$
24-hour	62	2	260 $\mu\text{g}/\text{m}^3$
8-hour	150	5	300 $\mu\text{g}/\text{m}^3$
15-minute	300	9	5000 $\mu\text{g}/\text{m}^3$
Coke Ovens			
Annual	10	4.1	75 $\mu\text{g}/\text{m}^3$
24-hour	115	47	260 $\mu\text{g}/\text{m}^3$
8-hour	300	123	300 $\mu\text{g}/\text{m}^3$
15-minute	500	205	5000 $\mu\text{g}/\text{m}^3$

Table 3. Summary of Ambient Concentrations and Exposure Levels of Concern

Averaging Period	Modeled Mn Concentration ($\mu\text{g}/\text{m}^3$)	Monitored TSP ¹ Concentration ($\mu\text{g}/\text{m}^3$)	Comments
15-Minute	250	N.A. ²	In order to protect against manganism, ACGIH recommends occupational workplace levels do not exceed 5000 $\mu\text{g Mn}/\text{m}^3$ for any 15-minute period.
8-Hour	125	N.A. ²	In order to protect against manganism, WHO recommends that occupational workplace levels not exceed 300 $\mu\text{g Mn}/\text{m}^3$ for any 8-hour time weighted averaging time.
24-Hour	47	144 ³	The primary 24-hour NAAQS for particulate matter established to protect public health is 260 $\mu\text{g}/\text{m}^3$.
Annual	4.1	52 ⁴	The primary annual NAAQS for particulate matter established to protect public health is 75 $\mu\text{g}/\text{m}^3$, geometric mean.

¹ Mn exists as particulate matter and is reported to comprise a fraction of total suspended particulate (TSP) matter that may range up to 25 percent.

² Not available.

³ Maximum monitored since 1981 at three ferroalloy facilities.

⁴ Annual geometric mean.

REFERENCES

American Iron and Steel Institute (1984). Annual Statistical Report.

Anderson, M.E., T.L. Curtin and M.G. Smith (1984). Regulatory Options Analysis for Manganese--Draft Final Report. EPA Contract No. 68-02-3599. Prepared for the Pollutant Assessment Branch, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

EPA [U.S. Environmental Protection Agency] (1982). Review of the National Ambient Air Quality Standards for Particulate Matter: Assessment of Scientific and Technical Information (OAQPS Staff Paper), Office of Air Quality Planning and Standards, Research Triangle Park, N.C. EPA 450/5-82-001.

EPA (U.S. Environmental Protection Agency) (1984). Health Assessment Document for Manganese. Environmental Criteria and Assessment Office, Office of Research and Development. U.S. Environmental Protection Agency, Cincinnati, Ohio.

RADIAN (1984). Study of Sources of Chromium, Nickel and Manganese Air Emissions--Final Report. Radian Corporation. EPA Contract No. 68-02-3818. Prepared for the Pollutant Assessment Branch, Strategies and Air Standards Division, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

Systems Applications, Inc. (1983). Assessment of Human Exposure to Atmospheric Concentrations of Selected Chemicals. PB 84-102-540 and PB 83-265-249. EPA Contract No. 68-02-3066. Strategies and Air Standards Division, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina.

U.S. Department of the Interior, 1983 Minerals Yearbook.

TAB A

Summary Review of Health
Effects Associated with Manganese Exposure

I. Overview

- Manganese is an essential element in the diet of humans and many other mammals.
- The systemic absorption of ingested manganese (oral exposure route) is determined by a complicated homeostatic mechanism that maintains constant tissue levels.
- Excess ingested manganese, dietary as well as the majority of inhaled manganese (via lung clearance mechanisms) is excreted in the feces.
- The primary manganese compound of concern as an air pollutant is MnO_2 , which is not soluble in water; consequently, only a small amount of the MnO_2 inhaled is biologically available.
- The manganese that is absorbed into the blood stream from the lung can penetrate the blood brain barrier and elicit CNS effects.
- Increased manganese absorption has been demonstrated in iron-deficient individuals.

II. Manganese Toxicity - Oral Exposure

A. Animals

- The LD_{50} in rats for oral exposure to MnO_2 is 7,400 mg Mn/kg. For comparison, the LD_{50} in rats to table salt (oral exposure) is 3,000 mg/kg.

B. Humans

- Very rare and when reported is associated with soluble compounds such as potassium permanganate, which can cause gastric irritation and impaction at high doses as well as potassium imbalances.

III. Manganese Toxicity - Inhalation Exposure

A. Respiratory Effects

1. Animals

- Pulmonary congestion and inflammation, as well as increased susceptibility to infection, occurs in animals exposed to manganese compounds by intratracheal or inhalation exposure routes. The lowest concentration reported in the HAD to cause inflammatory changes in animal lungs was $700 \mu\text{g}/\text{m}^3$. This study involved only 2 monkeys exposed to MnO_2 dust 22 hours/day for 10 months. Three other monkeys were exposed to $3,000 \mu\text{g}/\text{m}^3$ for the same exposure period. Inflammatory changes occurred earlier in the high dose.

2. Humans

- The respiratory effects elicited by exposure to manganese dust are the same as for any other relatively inert particulate matter. The index effect and severity depends on concentration and duration of exposure. The effects include sneezing, lacrimation, increased sputum, decreased lung function and chronic bronchitis. A significant number of studies have been conducted using insoluble respirable particulate matter not necessarily containing manganese. Clearly, acute effects such as sneezing and lacrimation can be elicited in healthy individuals with 15-minute exposures to particulate levels of $5,000 \mu\text{g}/\text{m}^3$. Fifteen-minute exposures to $1,000 \mu\text{g}/\text{m}^3$ can produce these effects, but less consistently in any given group of individuals.

- Metal fume fever consisting of fever, chills, sweating, nausea and cough is a reversible occupational disease resulting from immediate exposure in confined occupational settings to any number of metal oxides fumes, including manganese oxide fumes. The fumes are confined to the immediate

welding area, quickly becoming particulate in form within 10 or 12 feet. This toxic syndrome is restricted to the work place.

- The HAD reports 5 human studies that looked for respiratory effects at various estimated concentration ranges of manganese particulate matter. Three of these studies involved occupationally-exposed populations. Two were non-occupational. One of the non-occupational studies involved Japanese school children exposed to emissions from a ferromanganese plant. The other involved town residents in a town containing a ferromanganese plant. The latter study was inconclusive, and the former study revealed increased prevalence of minor respiratory symptoms associated with high levels ($200 \text{ kg/Km}^2/\text{month}$) of settled dust containing manganese. Although averaging times are not stated it appears from the data that manganese concentrations within the plant were above $300 \text{ } \mu\text{g}/\text{m}^3$ and suspended dust 100 meters away from the plant was measured to be $299 \text{ } \mu\text{g}/\text{m}^3$ (the manganese concentration was $4.04 \text{ } \mu\text{g}/\text{m}^3$). Manganese exposure in the occupational studies ranged from an estimated $.05 \text{ } \mu\text{g}/\text{m}^3$ to $220 \text{ mg}/\text{m}^3$. The duration of exposure was often not stated but chronic occupational exposure was assumed. In one occupational study, the levels of exposure associated with increased prevalence of chronic bronchitis in occupationally exposed people was estimated to be between $.4 \text{ mg}/\text{m}^3$ and $16 \text{ mg}/\text{m}^3$; however, the increased prevalence was only observed in smokers. No increased prevalence was observed in non-smokers exposed to these levels. There was not any difference in other respiratory symptoms, such as increased sputum and wheezing, associated with Mn exposure. Older studies, reported in 1946 and 1961, indicated increased pneumonia in manganese miners and potassium permanganate workers. The dust concentrations in the mine ranged from $28 \text{ mg}/\text{m}^3$ to $840 \text{ mg}/\text{m}^3$, with manganese concentrations in the dust ranging from 2-200 $\text{mg Mn}/\text{m}^3$. Dust

concentrations in the potassium permanganate plant were not reported, but the range of Mn concentrations in the dust was between $.1 \text{ mg/m}^3$ and 13.7 mg/m^3 .

B. Neurotoxic Effects

1. Animals

- The HAD reports 11 studies designed to examine the neurotoxic effects of manganese in experimental animals. Only one of these used the inhalation route of exposure. In this study, rats and monkeys were exposed to Mn_3O_4 continually for 9 months at concentrations as high as $1,152 \text{ } \mu\text{g/m}^3$, with no CNS abnormalities observed. In one study, rabbits were inoculated intratracheally with 400 mg of MnO_2 as a single dose. After 24 months, these animals experienced paralysis of the hind limbs. Three of the studies used the parental route of exposure (subcutaneous and intramuscular injection) and elicited behavioral changes. The lowest dosage regime associated with behavior changes was 39.5 mg Mn/kg , one dose a week for 9 weeks.

2. Humans

- The HAD presents 13 epidemiology studies associating manganism (neurologic disorder) in humans with chronic occupational exposure to manganese from various industrial processes. Estimated exposure levels range from less than $.0007 \text{ } \mu\text{g/m}^3$ to 450 mg/m^3 . The study populations ranged from 369 workers in one study to 9 in another. Signs and symptoms reported ranged from none observed to severe manganism. One 1977 study involving a study population of 369 and estimating a range of manganese exposure of from $.30 \text{ mg/m}^3$ to 20.44 mg/m^3 , with 27% (99 workers) exposed less than 4 years and 9.8% (36 workers) exposed over 20 years, reported 62 workers showing signs of tremor at rest. Although not unique to manganese exposure,

tremor at rest is an early sign of manganism. The HAD also states that there is no clear evidence of chronic manganese poisoning at occupational exposures to under 5 mg/m³.

IV. Reproductive and Cardiovascular Effects

- Severe manganism has been associated with impotence in humans, but the HAD reports that manganese per se is not likely to influence reproductive parameters.

- At occupational exposures from 0.39-20.44 mg/m³, human studies have indicated decrease in systolic blood pressure. Animals fed 564 ppm Mn in their diet had increased blood levels of serotonin, which decreases systolic blood pressure. The significance of this finding is not discussed in the Health Assessment Document for Manganese.

V. Perspectives

- The two major clinical manifestations of manganese exposure are not a single biological endpoint but are relatively progressive given sufficient dose and duration of exposure. The clinical manifestations of inhalation exposure to manganese as a component of fine ($\leq 2.5 \mu\text{m}$) particulate matter is identical to effects observed for any composite particulate matter and progress from increased minor respiratory effects (tearing, sneezing, increased sputum, decreased pulmonary efficiency) to increased susceptibility to infections and chronic bronchitis. Given sufficient manganese deposition in the lung over time to allow absorption into the blood stream and transport to the brain, neurologic symptoms ranging from tremor and psychotic behavior to actual brain damage will occur, again dependent on level of concentration in the brain. Depending on the severity of the effect and duration of severe effects, the symptoms are generally

reversible once exposure is terminated. Residual damage and recovery time are also dependent on severity of the effect over time.

- The probability of correctly predicting that a given effect will occur over a given time period at a given concentration by a particular route of exposure is a function of the number and quality of the studies available. In other words, the strength of the data is a function of the repeatability and predicting that a given effect will occur at a given concentration using one study has a low probability of being correct.

- Occupational epidemiology studies result in data showing a ratio or percentage of workers affected in a study population all of which are exposed to an estimated range of concentrations. There is a high probability that affected workers were exposed to the concentrations in the upper end of the range rather than the lower. Epidemiology studies are crude screening tools and by themselves seldom, if ever, provide clean-cut causal relationships. However, in the case of manganese, the number of occupational studies available although not of high quality by current standards clearly associate manganism in populations chronically exposed in occupational settings to concentrations of manganese above $5000 \mu\text{g Mn/m}^3$. Similarly, the total suspended particulate levels associated with such high concentrations of manganese have the potential of eliciting respiratory effects in exposed populations. Biological variability precludes identifying the lowest particulate level that can be associated with any defined respiratory effect; however, most mammals can be subjected to levels around $5000 \mu\text{g/m}^3$ for several hours without any serious adverse respiratory effects. It can

be noted, however, that the National Ambient Air Quality Standards for Particulate Matter¹ have been established to protect the public health with an adequate margin of safety.

¹ The Primary National Ambient Air Quality Standards are 260 $\mu\text{g}/\text{m}^3$, 24-hour average not to be exceeded more than once a year and 75 $\mu\text{g}/\text{m}^3$, annual geometric mean.

TAB B

Particulate Matter Concentrations in the
Vicinity of Active Ferroalloy Facilities

The short-term dispersion modeling results indicated that the ferroalloy industry was associated with the highest 8-hour and 15-minute modeled concentrations of manganese. In order to further analyze these results, monitored data were acquired from telephone conversations with district engineers who were familiar with the specific ferroalloy plants producing ferromanganese and silicomanganese in the U.S. in 1983. Information available in the 1983 Minerals Yearbook indicates that the ferromanganese and silicomanganese industry is a struggling domestic industry with production limited to five companies.

Production of ferromanganese in 1983 was only 64.2 metric tons down from 144 metric tons in 1981. The production of silicomanganese was so concentrated in 1983 that the data were not publishable. Only one of the five plants manufactured silicomanganese exclusively; two others produced both ferromanganese and silicomanganese and the other two manufactured ferromanganese only. Telephone calls were subsequently made to the State district engineers familiar with the actual plants. Three of the plants had TSP monitors within five miles of the plant. All of the plants are subject to State mandated emission regulations. The two plants not having TSP monitors within five miles of the plant were described by the district

engineers as "a pretty clean plant with ESP and a scrubber set up" and "complies with State emission regulations for particulate matter," respectively.

The monitored data available for the other three plants revealed that the maximum 24-hour average particulate matter concentration measured since 1981 was $144 \mu\text{g}/\text{m}^3$ (1982) and the highest annual geometric mean $52 \mu\text{g}/\text{m}^3$ (1984). All measured levels available since 1981 indicate that the NAAQS for particulate matter are being attained in the vicinity of the existing ferroalloy plants.



TAB C

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

February 21, 1985

MEMORANDUM

SUBJECT: Screening Procedure for Estimating Short-Term Air Concentrations from Toxic Releases

FROM: Johnnie L. Pearson, Chief
Model Application Section (MD-14)

A handwritten signature in dark ink, appearing to be "Johnnie L. Pearson", written over the "FROM:" line.

TO: Robert M. Schell, Chief
Program Integration and Health Section (MD-12)

The purpose of this memorandum is to describe the screening procedure we have used to date in order to estimate short-term air concentrations to assist in your assessment of potential acute health effects associated with airborne toxic compounds. The procedure is intended to be conservative. In other words, the estimates, normalized by the emission rates, may be regarded as being in the upper range of likely values. This is accomplished by examining worst case meteorological conditions and by incorporating a safety factor to account for the inherent uncertainty in the calculations. It is important to note, however, that unless maximum short-term emission rates are considered in the procedure, the resulting concentration estimates cannot be regarded as conservative. Indeed, intermittent releases of short duration could result in concentrations several orders of magnitude higher than constant releases of long duration having the same average annual emissions.

The screening procedure follows the approach outlined in the document "Procedures for Evaluating Air Quality Impact of New Stationary Sources," EPA-450/4-77-001, commonly referred to as Volume 10 of the AQMPA guideline series. This approach defines a variety of potential worst case meteorological or dispersion scenarios which are expected to encompass the particular worst-case scenario for the source in question. Six scenarios are recognized in the guideline. These are referred to by their descriptive names as: (1) looping plume; (2) limited mixing; (3) coning plume; (4) fanning plume; (5) fumigation; and (6) downwash. In short, 1-hour average concentrations are computed for each of these scenarios for which a reasonable possibility exists that high ambient air concentrations for the source in question could result. The calculations involve a flat terrain assumption and are essentially equivalent to those made in such screening models as PTPLU, PTMAX, or PTDIS. For a true ground-level source, ground-level concentrations decrease monotonically with distance. For these and other near ground-level releases, the calculations were performed for a downwind

distance of 200 meters. The implicit assumption here is that the plant property extends to a distance of at least 200 meters (650 feet) from the source.

For sources where building downwash is a potential concern, the screening calculation involves an estimate of the air concentrations in the cavity of the building wake region under steady state, worst case conditions. In general, such concentrations are not expected to extend to a distance of 200 meters downwind of the source. Should future analyses indicate that the building downwash scenario is of potential concern in terms of ambient concentrations, consideration might be given to the development of an alternative screening approach.

The Volume 10 procedure also considers concentrations at specified receptors, for example on elevated terrain. For many elevated releases, plume impaction on elevated terrain can lead to high ground-level concentrations. However, the occurrence of plume impaction is highly dependent on the particular source characteristics in relation to the surrounding terrain. Obviously, this is highly site-specific. Nevertheless, a suitable screening procedure must consider the possibility of plume impaction since toxic releases are not limited to facilities located in flat terrain.

In the absence of a site-specific analysis, the screening procedure assumes plume impaction can occur at specified distances, depending on the effective stack height, H . For H less than 50 meters, the impaction distance is taken as 500 meters, for H between 50 and 100 meters as 1000 meters, and H greater than 100 meters as 2000 meters. Since the Volume 10 procedure considers only 24-hour averaging times for the plume impaction scenario, it is necessary to modify the approach to estimate short-term concentrations, on the order of an hour. For this purpose, a COMPLEX II type of calculation is made, i.e., no sector averaging is used. Specifically, a 1-hour plume centerline concentration is calculated, assuming full ground reflection, under conditions of F stability and 2 m/s winds, at the specified impaction distance. While these dispersion-related assumptions may be regarded as quite conservative, the specified impaction distances are not necessarily conservative in all cases. In cases where plume impaction could occur at shorter distances, the degree of conservatism in the overall concentration estimate would be reduced.

It should be noted that the plume impaction screening calculation does not consider other atmospheric conditions that are known to cause high ground-level concentrations in complex terrain in specific situations. For example, sources located on the lee side of major terrain features may be subject to downdrafts which can lead to greatly increased ground-level concentrations. Also not considered explicitly is valley stagnation, a situation in which cold air becomes trapped in a valley for an extended period, leading to a build-up of concentrations over time.

The Benefits of HiTEC 3000 on Reducing Urban Smog
Could be Even More Significant

Ethyl Corporation's HiTEC 3000 performance additive could show even greater benefits to efforts to reduce urban smog based on a new National Research Council report on ozone. The National Research Council (NRC) of the National Academy of Sciences has recently released a study which strongly recommends that nitrogen oxides must be reduced in order to control ozone formation. Ozone, or urban smog, is formed when volatile organic compounds (VOCs) and nitrogen oxides (NO_x) react in the atmosphere in the presence of sunlight. For many years, the control strategy for ozone has focused on reducing VOCs. The report notes that for years officials have underestimated the amount of manmade volatile organic compounds and have not included the significant sources of natural (from plants or trees) volatile organics. Because the ratio of VOCs/ NO_x is critical for formation of ozone, this underestimation of VOCs has resulted in an overestimate of the effectiveness of controlling VOCs and underestimated the value of controlling NO_x in ozone control efforts. Thus, in spite of spending billions of dollars for control efforts, ozone levels have decreased only slightly or not at all. The NRC report, entitled "Rethinking the Ozone Problem in Urban and Regional Air Pollution," emphasizes the necessity to reduce nitrogen oxides.

The report states that automobiles and trucks account for 45% of the anthropogenic (manmade) volatile organic compounds, 50% of the nitrogen oxides, and 90% of the carbon monoxide in cities where the national ambient air quality standard for ozone is not met.

The results of Ethyl Corporation's 48-car fleet test showed that use of HiTEC 3000 performance additive reduced nitrogen oxides an average of 20 percent (0.11 grams/mile) over the 75,000 mile period of the study. Applying these test results to the existing national car fleet on the road, this reduction would, over a short time period, amount to a 10 percent reduction in total oxides of nitrogen from all sources, which represents a very substantial reduction.

As part of Ethyl's waiver effort, urban air shed modeling for ozone was carried out for two cities, Philadelphia and Atlanta. The results showed that the introduction of HiTEC 3000 in 1991 into all light duty gasoline vehicles would reduce ozone equivalent to removing 129,000 (7%) of gasoline vehicles from the road in Atlanta and 170,000 (5%) of the gasoline vehicles from the road in Philadelphia. By considering the concerns raised by the NRC report, such as emphasis on NO_x control, overestimation of control of manmade VOCs and no consideration of natural VOCs, the impact of the use of HiTEC 3000 would be even more impressive in ozone reduction efforts.

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ATTACHMENT 2

The procedure described above results in a set of 1-hour concentrations. These are doubled to provide an explicit margin of safety to account for uncertainties in the dispersion calculations. This does not account for uncertainty in the source characterization, however.

For comparison with ACGIH threshold limit values, or TLV's, the 1-hour averages are adjusted using a power law relationship. In effect, the 1-hour estimates are increased by a factor of 1.3 to obtain a nominal 15-minute value for comparison with the STEL value. The 1-hour estimates are decreased by a factor of 0.7 to obtain a nominal 8-hour value for comparison with the TWA value. In general, these adjustments are most appropriate for near ground-level sources. In particular, they may not be appropriate for elevated releases in complex terrain. Further consideration of this aspect is warranted. Mass concentrations were converted to a volume basis, i.e., ppm, assuming standard conditions of temperature and pressure (25°C and 760 mm Hg).

In cases where multiple sources may be present at one location, the individual source contributions are summed for each of the scenarios described previously in order to find the maximum concentration from all sources combined. In this procedure, the total combined impact on ambient air is not determined by combining different scenarios since unrealistically large concentrations could result.

Please note that the above screening procedure is inappropriate for large area sources. For example, concentrations 200 meters downwind from a 200 meter square area source could be overestimated by as much as a factor of 20 or more. Where large area sources are a concern, modifications to the screening procedure can be made to account for the initial dilution of the emissions over the area of the source. In analyses to date, this appears not to have been a factor.

The above should adequately summarize the dispersion methodology. If you have further questions, please contact Dave Layland at extension 5690.

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